PB83-250357

Gas Stove Usage, CO and TSP, and Respiratory Effects

Arizona Univ. Health Sciences Center, Tucson

Prepared for

Health Effects Research Lab.
Research Triangle Park, NC

1983

U.S. Department of Commerce
National Technical Information Service
NTIS

PB83-250357

EPA-600/D-83-107
August 1983

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by

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EPA Grant R805318

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TECHNICAL REPORT DATA
(Please read Instructions on the reverse before completing)

1. REPORT NO.	2.	3. RECIPIENT'S ACCESSION NO.
EPA-600/D-83-107		PB83 250357

4. TITLE AND SUBTITLE	5. REPORT DATE
Gas Stove Usage, CO and TSP, and Respiratory Effects	1983
	6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)	8. PERFORMING ORGANIZATION REPORT NO.
M.D. Lebowitz, G. Corman, C.J. Holberg, R. Dodge, M.K. O'Rourke	

9. PERFORMING ORGANIZATION NAME AND ADDRESS	10. PROGRAM ELEMENT NO.
Div. of Resp. Sciences, U. of Arizona Health Sciences Center, Tucson, AZ	AAGA1D
	11. CONTRACT/GRANT NO. R805318

12. SPONSORING AGENCY NAME AND ADDRESS	13. TYPE OF REPORT AND PERIOD COVERED
Health Effects Research Laboratory Office of Research and Development U.S. Environmental Protection Agency Research Triangle Park, NC 27711	14. SPONSORING AGENCY CODE EPA-600/11

15. SUPPLEMENTARY NOTES
P.O. Carl Hayes

16. ABSTRACT

A symptom stratified, geographic cluster sample of 117 middleclass households was studied. Symptom daily diaries were obtained for 211 subjects for 2 years. Indoor sampling in 41 house was for O_3, TSP, RSP, CO, temperature (T), and relative humidity (RH). Questionnaires determined type of stove and smokers in all households. Ambient pollutants (O_3, TSP, CO, NO_2), were monitored in or near the clusters, as was T, RH. Smoking in the household was significantly correlated with TSP and RSP. Indoor CO significantly correlated with gas stove usage, but not with outdoor CO. Children's Vmax was associated only with outdoor factors. Asthmatic Vmax was associated with smoking, gas stove use, NO_2, and occasionally O_3, T, and RH. Indoor (not outdoor) TSP was significantly correlated with rhinitis (Rh) and asthma attacks in asthmatics (AS), eye irritation (E), and productive cough (PCS). Indoor CO (not outdoor) was significantly correlated with Rh and E. Using analysis of variance, controlling for age, sex, smoking and ambient environmental variables, gas stoves were significantly associated with increased prevalence rates of Rh in asthmatics and non-asthmatics, sore throat in non-asthmatics, and PCS in asthmatics and allergics. Ambient NO_2 was associated with E and PCS in allergics. Micro-pollen was associated with E, Rh and AS.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group

18. DISTRIBUTION STATEMENT	19. SECURITY CLASS (This Report)	21. NO. OF PAGES
Release to Public	Unclassified	16
	20. SECURITY CLASS (This page) Unclassified	22. PRICE

EPA Form 2220-1 (Rev. 4-77) PREVIOUS EDITION IS OBSOLETE

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This work was supported by EPA Grant #R805318 and NIH SCOR Grant #HL14136.

"Although the research described in this article has been funded wholly or in part by the United States Environmental Proctection Agency through contract or grant #R805318 to Michael D. Lebowitz, Ph.D., it has not been subjected to the Agency's required peer and policy review and therefore does not necessarily reflect the views of the Agency and no official endorsement should be inferred."

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INTRODUCTION

Unvented appliance combustion, especially those using some form of gas, can produce indoor concentrations of carbon monoxide and oxides of nitrogen, which frequently exceeds of acceptable occupational air quality standards. Studies have shown striking differences of NO_2 levels in homes with gas as compared to electric stoves (Dockery et al., 1981; Yocom, 1982). These studies demonstrated that levels are dependent on combustion efficiency and ventilation. Indoor concentrations are higher than those outdoors (Moschandreas et al., 1978; NRC, 1981), depending on activity levels. These pollutants also come from other activity-related sources, especially sidestream smoke, although the impact of environmental tobacco on indoor concentrations of NO_2 is usually small compared to the impact of gas stoves (Yocom, 1982). In addition, residential wood and coal stoves are potential sources of carbon monoxide and oxides of nitrogen as well as respirable particles (NRC, 1981).

Previous studies have explored associated respiratory symptoms; they have been somewhat contradictory (Melia et al., 1979; Keller et al, 1979a-b; Florey et al., 1979; NRC, 1981). The amount of carbon monoxide from indoor combustion sources can be shown to produce increases in carboxyhemoglobin. Blood carbon monoxide levels may be higher in individuals exposed to passive smoke than in those non-exposed (Olshansky, 1982; Radford et al., 1982), but are probably lower than the carboxyhemoglobin levels associated with exposures to unvented gas stoves (NRC, 1981). The effects of CO have been well defined by many other studies (NRC, 1977), but the health implications of indoor CO levels remain speculative. A major concern is that the carboxyhemoglobin and methemoglobin (from NO_2 and NO) accumulate from exposure to multiple sources and act in an additive fashion as adverse risks to health (Case et al., 1979). Some respiratory diseases may occur in indoor situations in which high levels of respiratory particles and gases are due to wood burning (Anderson, 1979; Sofoluwe, 1968). As with other pollutants, sensitive populations, such as asthmatics, are of major concern.

Recently, studies of the effects of gas stove usage have been performed by several groups (Comstock et al., 1981; Dodge, 1982; Melia et al., 1982; Speizer et al., 1980a-b) where passive smoking has been studied as well. The results have been quite contradictory. Speizer et al., (1980a-b) demonstrated the effects of gas stoves, but not effects of environmental tobacco smoking (a significant reverse trend was seen). However, there may be possible effects of passive smoking in the same study with different analyses of further data (Dockery et al., 1982). Dodge (1982) did not find any effect of gas stove usage or passive smoking when examining children's lung function, cross sectionally or longitudinally, though self reported cough was higher in children exposed to parental smoking. Comstock et al. (1981) examined environmental tobacco smoking in adults, and gas cooking; they only found an effect of gas cooking in males. Studies by Binder et al.

(1976) and Schilling et al. (1977) utilizing indoor measurements showed that indoor levels of respirable particles were higher in homes with smokers, but that there was no effect on children's symptoms or lung function.

This paper will examine daily peak flows in asthmatic and non-asthmatic families in association with exposures to gas stove emissions and passive smoking. Pollutant concentrations are examined, as are interactions and confounding factors.

METHODS

We studied 117 families (229 subjects) in Tucson, which were derived from a stratified sample of families in geographic clusters from a representative community population under study (Lebowitz et al., 1975). The families studied were essentially all middle class and represented families with and without reported asthma. They have been monitored over a two-year period, using daily diaries for symptom information and peak flows. Peak flows were performed between 1500-1900 hours each day using a mini-Wright peak flow meter (Lebowitz et al., 1982a). The subjects were trained in the use of the peak flow meters and symptom diaries by trained nurses. Monthly checks by phone and seasonal checks by visit were made to insure proper usage. The mini-Wright peak flow meters were calibrated monthly. Diary response rates were acceptable for a majority of days in all seasons. All families provided information as to characteristics of the house, yard and streets, heating, cooling, type of stove, other appliances and smoking in the household.

Micro-indoor and outdoor monitoring have been conducted in a random cluster sample representative of all study households (n = 41) for temperature, humidity and air pollutants (pollen, bacilli, fungi, and algae), for 72 hours each. Macro-monitoring for air pollutants (and pollen) was conducted simultaneously near the center of each cluster.

Indoor samples were measured in living rooms and kitchens. Particulate was measured using Gelman filters attached to a Thomas pump (1.7 L/m) for total suspended particulate (TSP), and with a cyclone fractionator for respirable suspended particulate (RSP); identical units were used outside the house (one in front and one in back). CO was measured using grab bag samples and a Bendix CO IR analyzer. NO_x measurements using Bendix chemiluminescent analyzers from EPA were unsuccessful. O_3 measurements used a Daisibi chemiluminescent monitor. Micro-environmental sampling of pollen was conducted using Rotorod Samplers and macro sampling was done utilizing a Burkard 7-day volumetric pollen samplers (O'Rourke, 1980). The clustering of plant types on the micro- and macro-levels have been examined previously (O'Rourke, 1982), as has relationships of pollen. Micro-sampling of fungi and algae has been described elsewhere (Lebowitz et al., 1982b). Daily macromonitoring of air pollutants was performed routinely by the Pima County Health Department O_3, CO, NO_2, and TSP (every sixth day). Meteorological information was obtained from the U.S. Weather Bureau.

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Symptom rates per 100 person days were calculated from daily diary data for asthmatics and non-asthmatics within exposure groups, for comparative purposes. Peak flows were transformed to standard normal deviates (z-scores) to remove effects of height, age, and sex. Differences in the number of observations per subject were adjusted by using the number as a co-variate. Statistical techniques used were frequency distributions contingency tables and multivariate analysis of variance, using SPSS programs on a DEC 10-Cyber 175 computer system.

RESULTS

Indoor pollutant levels:

Indoor TSP ranged from 5.7 to 68.5 $\mu g/m^3$. Simultaneous micro-outdoor TSP ranged from 2.1 to 169.6 $\mu g/m^3$. Cyclone measurements of RSP were often below readable levels; the maximum was 49.7 $\mu g/m^3$, indoors and 124.5 $\mu g/m^3$ outdoors. Electron microscopy (EM) could not identify the type of dust found on house floors, except that it differed from outdoor TSP. Total micro-outdoor pollen counts were approximately 5% of the macro-pollen counts (max = 1517), and micro-indoor pollen counts were only 5% of the micro-outdoor pollen counts. Floor samples contained high amounts of nonsuspended pollen in the spring (up to 5.5 x 10^6 grains/gm) as determined by light microscopy; although none were seen by EM. The common pollen in this region usually had Gaussian distributions of mass median aerodynamic diameter (MMAD), ranging from 17 to 36 µm. The minimum MMAD for common pollen types were generally between 10 and 15 µm.

Averages of the two indoor spot CO readings were similar and were lower than 2.4 ppm; micro-outdoor readings were <3.8 ppm. These were lower than those reported elsewhere, and well below both threshold limit values (TLV) and National Ambient Air Quality Standards (NAAQS) (NRC, 1981). Indoor O_3 concentrations were between 0-0.035 ppm, which is as low as found elsewhere (NRC, 1981) and well below TLV and NAAQS. Temperature and humidity varied by season, but the former did not vary very much indoors over the year and the latter did not differ much between indoors and outdoors over the year. Algae found outside or in evaporative coolers were not found indoors. Fungi were either ubiquitous or only found in one location (indoors, outdoors, or in coolers.

Correlations of indoor and outdoor pollutants:

Indoor CO was correlated significantly with micro-outdoor CO ($r=.60$, $p<.003$) but not with regional CO measurements (1.5 - 4.9 ppm on days of indoor monitoring). Indoor CO was significantly associated with gas stove usage, but not with environmental tobacco smoke (Table 1), nor with gas burning furnaces, hot water heaters or washers - dryers (ie, vented appliances). Thus, though low, CO was used as the indicator pollutant measure of combustion emission from the gas stoves, because NO_2 concentrations were unavailable. There was no temporal trend of indoor or micro-outdoor CO from house to house, though there was a temporal trend of regional CO levels.

Indoor TSP was significantly correlated with micro-outdoor TSP ($r=.71$, $p<.001$) and regional TSP ($r=.4$, $p<.05$); the latter ranged from 27.5 - 129 $\mu g/m^3$ on days of indoor monitoring. Indoor RSP was significantly correlated with micro-outdoor RSP ($r=.77$, $p<.001$). Indoor and micro-outdoor TSP and RSP were higher when all outside streets around the house were dirt (rather than paved, but not when only the "back alley" was unpaved). Measurements showed about a 10-fold fall of outdoor TSP within 5 feet of the front door and about a 100-200-fold fall of outdoor TSP in the middle of the living room. Both TSP and RSP were significantly associated with household smoking, but not with gas stove usage (Table 1). Indoor TSP and RSP showed no temporal trends, although regional TSP has a daily trend.

Neither gas stove usage nor household smoking was associated with social status in this sample. Attempts to estimate concentrations of CO, TSP, and RSP in houses not monitored, by cluster and season, essentially yielded a classification based on gas stove usage and smoking in the houses.

There were 24 children who had daily peak flows ($\dot{V}max$) over an 11-month period. No inter-subject differences in baseline data were seen after age and height correction; all were within the normal range. Temperature had been shown to be significantly inversely related to $\dot{V}max$, also in conjunction with outdoor O_3. $\dot{V}max$ was positively correlated with relative humidity RH ($p<.001$) and negatively with precipitation (P) ($p<.025$). Outdoor O_3 and TSP had been independently significantly related to $\dot{V}max$, after controlling for outdoor NO_2, CO and season. In analysis of indoor and outdoor variables, the indoor explanatory variables were gas stove usage and passive smoking. There was a significant association of $\dot{V}max$ with the interaction of TSP and O_3. O_3 has a relation to $\dot{V}max$ in those not exposed to passive smoking; those with passive smoking had a non-significantly higher average $\dot{V}max$. The effect of outdoor environmental factors on those young subjects $\dot{V}max$ is probably related to the amount of time spent outdoors and exercise (Lebowitz et al., 1974).

$\dot{V}max$ in adults was related to environmental conditions differently in different symptom and smoking groups. Active and previously active smoking negatively affected $\dot{V}max$ in all groups. The multivariate analyses of variance yielded associations of $\dot{V}max$ with gas stove usage in asthmatics, normals and allergics, outdoor NO_2 in the same groups, outdoor CO in asthmatics and allergics, outdoor O_3 in those with symptoms of non-asthmatic airways obstruction (AOD) and with temperature in asthmatics. High relative humidity affected $\dot{V}max$ in AOD. Season affected various groups as well (related to combinations of meteorological conditions, air pollutants, and pollen). There was probably a correlation of indoor and outdoor NO_2, as previously reported by others (NRC, 1981), the indoor NO_2 associated with gas stove emissions. In this study, there was an independent interactive effect of NO_2 and gas stove usage on $\dot{V}max$ in asthmatics, as well as an independent effect of gas stove usage, as shown in Table 2. Active smoking and gas stove usage in asthmatics combined to decrease $\dot{V}max$ as well.

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In asthmatics in one cluster (all asthmatic families), there were additional independent associations with $\dot{V}max$: high relative humidity (as a co-variable), and explanatory variables: micro-pollen, active smoking, and indoor TSP (as seen in Table 3). Neither passive smoking nor fungi had any significant effects (on $\dot{V}max$ or symptoms). Indoor RSP did not have a significant association with $\dot{V}max$ in this group or other groups. Low temperature had a significant effect on $\dot{V}max$ in asthmatics in this cluster, and on "asthma attacks" as well; the effect was weak for asthma attacks once all household factors were included (in which dirt roads (TSP) and pets were significantly related (positively) to asthma attacks. (Correct classification of attacks was 84% by discriminant analysis.) Passive smoking, evaporative coolers and other appliances were not associated with any daily symptom prevalence rates in adults. However, indoor TSP and RSP (but not outdoor TSP) were associated with increased "asthma" symptoms in asthmatics and rhinitis in asthmatics (though weakly along with smoking and low temperature) and non-asthmatics. Indoor TSP was associated also with eye irritation in all adults and increased productive cough in non-asthmatics.

Gas stove usage was associated also with increased rhinitis in all adults (as was indoor CO), increased productive cough in asthmatics and allergics, and increased sore throat prevalence rates in non-asthmatics. Indoor CO was associated with eye irritation. Age and sex adjusted "asthma attacks" in adult asthmatics, as mentioned, occurred most often in cold temperatures; they may or may not be associated with gas stove usage, but they definitely were not associated with outdoor gaseous pollutants (except some minor, complex interaction of O_3 with temperature). ("Asthma" symptoms were not day-of-week dependent.) Micro-pollen was associated with "asthma" symptoms, ~~as well~~ as eye irritation, and rhinitis (in summer).

DISCUSSION

Indoor TSP had an effect on adults' daily symptoms and on asthmatics' $\dot{V}max$, which must be associated with other characteristics of the indoor TSP (as it is correlated with environmental tobacco smoke). Unfortunately, because of sample size and the number of confounding factors, a precise exposure-effect relationship could not be determined. This would appear to be a major methodological problem for these types of studies, as one must account for co-variables (age, sex, smoking), symptoms (asthma, allergy, AOD), indoor and outdoor meteorological, pollutant and pollen differences.

The effect of gas stove emissions was more pronounced than expected. Minor perturbations of effects from gaseous pollutants from other sources may be hidden. One could estimate that effects would be seen from environmental tobacco smoke if it were 3-5 times greater (the approximate degree that stove emissions exceed sidestream smoke (NRC, 1981) or if it were 9 times greater, based on dose estimates of sidestream smoke (Hiller et al., 1982), to see the same effects as active smoking.

There were no effects of passive smoking discernable on $\dot{V}max$ or symptoms in any group, asthmatics or others. This was similar to the negative findings for children's lung function in a study of the 344 nuclear families in the larger community population (Lebowitz et al., 1982a), from which this study sample was drawn. In the larger study, no effects of environmental tobacco smoke on respiratory symptoms or past respiratory illness in the 700+ children and 3100 adults were found (Lebowitz and Burrows, 1976). This latter study showed more symptoms, including infection-type, in parents with symptoms, especially smokers.

Children under 25 and asthmatics both appeared more sensitive to the effects of various pollutants. Associations were seen for children's $\dot{V}max$ and for asthmatic adults' $\dot{V}max$, as well as for some symptoms in adults, especially asthmatics. Even outdoor conditions affected these groups. Methacholine challenges in 53 of the adult subjects demonstrated significant reactivity in asthmatics and intermediate reactivity in most with "asthmatic-type" symptoms; some without symptoms also showed intermediate reactivity. In addition to some validation of asthma in this study by objective tests, validation of Type I hypersensitivity is necessary as well; we used skin tests (for allergens) and measurements of circulating IgE.

The examination of the effects of various indoor and outdoor environmental conditions was of great benefit. Proper integrated quantitative exposure estimates would be of even greater benefit (WHO, 1982). Though not a critical variable in this study, daily occupational studies may have acute, and even chronic effects (Lebowitz, 1977). Peak exposure information would be useful also. (Unfortunately, the present study did not have this type of information.)

Based on levels of exposures the effects seen were probably more acute, reversible types, . However, it is unknown how frequent or severe such acute/reversible effects are required to produce chronic reversible or irreversible effects.

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TABLE 1

TSP, RSP AND CO BY ENVIRONMENTAL TOBACCO SMOKE

AND GAS STOVE USAGE

		No Smokers		Smokers		Smoking		Stove	
		Gas stove	Electric	Gas Stove	Electric	No	Yes	Gas	Electric
TSP $\mu g/m^3$	$\bar{x}$	19.3+	14.9***	30.6+	36.3***	17.7***	33.0	25.5	28.0
	s	11.2	8.9	20.0	14.9	10.7	18.2	17.6	16.6
	n	9	5	11	8	14	19	20	13
RSP $\mu g/m^3$	$\bar{x}$	8.7	4.2**	8.3	19.6**	7.2+	13.1	8.5	13.7
	s	8.9	4.6	7.5	15.9	8.0	13.1	8.2	14.8
	n	10	5	11	8	15	19	21	13
CO ppm	$\bar{x}$	1.4	0.9*	1.4*	1.0	1.2	1.2	1.41**	0.97
	s	0.6	0.4	0.6	0.6	0.6	0.6	0.56	0.54
	n	9	5	6	6	14	12	15	11

* Significant by 2 tailed t-test, $p<.05$
** $p<.025$
*** $p<.005$
+ borderline significant (2 tailed t-test) $.05<p<.10$

TABLE 2

INTERACTIVE EFFECT OF GAS STOVE USAGE AND OUTDOOR NO_2 ON ADULT ASTHMATIC AVERAGE $\dot{V}max$

	Stove	
NO_2 (ppm)	Gas	Electric
≤.0265	-1.54*	-1.24*
.027 - .0385	-1.53	-1.51
.039+	-1.53	-1.65*
Any	-2.16**	-0.76

ANOVA $p<.0001$

* t-test significant, $p<.01$

** $p<.0001$

TABLE 3

ASSOCIATIONS OF SMOKING AND INDOOR TSP WITH AVERAGE $\dot{V}max$ IN ADULT ASTHMATICS

(in asthma cluster)

	TSP ($\mu g/m^3$)			Smoking			
	≤25	25-70	All	Current	Ex	Never	All
$\bar{x}$	-1.17	-1.92	-1.56	-2.41	-2.18	-1.25	-1.77
s	.99	1.83	1.54	2.08	1.86	1.44	1.81
Person days	1205	1334	2539	857	687	1574	3118
ANOVA:	$p<.0001$				$p<.0001$		

	Micro-Pollen	
	Low-Mod	High
$\bar{x}$	-1.18	-2.51
s	1.78	1.73
Person-days	1027	533

ANOVA: $p<.0001$